THE TAMIL NADU Dr. MGR MEDICAL UNIVERSITY

CHENNAI –TAMIL NADU

DYSLIPIDEMIA IN PERITONEAL DIALYSIS RELATION TO DIALYTIC VARIABLES

An Evaluation

SUBMITTED FOR THE MD DEGREE EXAMINATION

BRANCH I

(GENERAL MEDICINE)

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THANJAVUR MEDICAL COLLEGE

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Introduction:

End stage renal disease (ESRD) represents a clinical state or condition in which there has been an irreversible loss of endogenous renal function, of a degree sufficient to render the patient permanently dependent upon renal replacement therapy in order to avoid life-threatening uraemia. Patients with ESRD have decreased quality of life, high morbidity and an annual mortality of about 22%. Renal replacement therapy (RRT) is required when kidney function deteriorates to the point where the accumulation of the waste products begins to interfere with life function.

Peritoneal dialysis, haemodialysis and renal transplantation are the major modalities of renal replacement therapy. The number of the patients kept alive by dialysis therapy continues to increase each year. The morbidity and mortality in the patients under going dialysis is a rapidly emerging problem. Dialysis is a procedure, which serves as a bridge to renal transplantation. . There are many studies going on to analyze the merits and demerits of the dialysis procedure.

We did this study to analyze one of the demerits of the peritoneal dialysis, ***hyperlipidaemias*** in the patients under going the peritoneal dialysis.

In our hospital we have a large number of chronic renal failure patients, who fulfilled the criteria for undergoing the dialysis; this study was conducted in such patients with their full co-operation.

Aim of the study

1. To estimate the various level of lipid abnormalities in the chronic kidney disease patients under going peritoneal dialysis.

2. To estimate which lipid abnormalities is more common in the study group.
3. To study, if correlation exists between the peritoneal dialysis and the levels of the atherogenic lipids.
4. To estimate, whether the abnormal lipids induced by peritoneal dialysis shows any correlation with the cardiovascular disease.

Inclusion criteria for the case selection:

1. Elevated renal parameters, with urea: creatinine ratio <20.

2. Reduced creatinine clearance < 30ml/min.

3. Contracted kidneys as evidenced by the ultrasound scan abdomen. Kidney Size of < 9.0 x4.0 cms.

4. History of treatment for Systemic hypertension for the minimum of two yrs duration, but not on chronic β blocker therapy.

5. Patient's general conditions to withstand the dialysis procedure for a continuous period of 48 hrs.

6. Age > 40yrs.

Exclusion criteria for the case selection:

1. Patients with known history of Diabetes Mellitus and who were not known diabetics with elevated random blood sugar values of >200mgs % was excluded
2. A known ischaemic heart disease patient on treatment. (Previous h/o myocardial infarction)
3. Severe co morbid conditions like pneumonia, alcoholic liver disease and hypotension
4. Patients on β blocker and thiazide diuretics for their hypertension during the study period

5. Patients with history of intake of the anti cholesterolemic agents
6. History of cigarette smoking
7. Any evidence of connective tissue disorder, particularly SLE
8. Bilateral renal artery stenosis, as a cause for the CKD
9. Patients with the features of hypothyroidism and obstructive liver disease

Materials and methods:

This study was conducted in THANJAVUR MEDICAL COLLEGE NEPHROLOGY department. By applying the inclusion and exclusion criteria we selected 41 patients for our study, among them 29 were male and 12 were female.

After proper history taking and the clinical examination, we did the basic investigations like, TC, DC, ESR, HB%, RBC count, and platelet count.

Estimating the blood sugar, urea, creatinine and serum electrolytes assessed the renal functions**.** We estimated the size of the kidneys, with ultrasound scan abdomen. (We had taken the longitudinal size of <9 cms and the cross sectional size of <4 cms as a cutoff value).

Screening for the ischemic heart disease were done with the help of electro cardiogram and the patients were excluded, who were all taking the ***anti ischemic therapy***, because this treatment alters the serum lipid levels.i.e:

Beta blockers – raises the serum LDL level with out altering the HDL level

Thiazide**s** ***diuretics*** raise the triglyceride level and reduce the HDL level.

We took the blood sample for lipid profile analysis before starting the peritoneal dialysis. (Pre Dialysis lipid profile sample)

We did the peritoneal Dialysis for the patients, who were all in the uraemic symptoms. Associated features present in these patients were

1. Hyperkalemia
2. Metabolic acidosis
3. Extra cellular fluid volume expansion

Procedure during dialysis:

Materials we were used for the dialysis,

1. Peritoneal dialysis catheter – straight Tenckhoff catheter.
2. Peritoneal Dialysis transfusion set.
3. Dialysate fluid.
4. Material for sterilizing the surface.
5. Suture material for fixing the catheter.

Composition of the Dialysate fluid, what we used for this study:

Each 100 ml contains

1. Magnesium chloride – 0.0152 gm
2. Calcium carbonate - 0.0220 gm
3. Sodium acetate - 0.4760 gm
4. Dextrose an hydrase – 1.7 gm
5. Sodium meta bisulphate -0.0150 gm
6. Water for injection.

Osmolality is approximately 355mosmol /l

Solution contains approximate mmol/l of

1. Sodium 130
2. Calcium 1.5
3. Magnesium 0.75
4. Chloride 100
5. Bicarbonate (as acetate) 35

With this above material we properly sterilize the abdomen with povidone ***iodine and spirit***, we made a small incision in the Abdomen in the sub umbilical region, we introduced the catheter through the sub-umbilical incision and positioned the catheter tube in the either one of the flanks. Drain was checked and the stay sutures applied for the catheter. Broad-spectrum antibiotics prescribed for the patient.

Duration of the dialysis: We subjected the patient to dialysis for continuous 48 hrs a period.

At the end of the dialysis procedure we sent the blood sample for the lipid profile analysis (post dialysis sample).

Additional investigations needed in this study:

1. 24hrs urinary protein estimation
2. creatinine clearance.

Procedure used to measure the **serum creatinine and creatinine clearance** – AUTO ANALYSER TECHNIQUE – using jaffe reaction.

Analyzing the Sensitivity and specificity of the investigations, what we used in this study.

We used **plasma urea, serum creatinine, creatinine clearence and ultrasound scan abdomen** were the necessary investigations to ***satisfy the criteria for the chronic kidney disease.***

1. Plasma urea:

Urea is one of the first indicators for the used to measure the renal function. **Plasma urea is a poor measure of the GFR**. Urea production is variable largely depends on the protein intake. Thus the kidneys excrete most of the urea [25,26]. It can be readily reabsorbed in the tubules. In conditions like low antidiuretic hormone levels, the medullary collecting tubules are impermeable to the urea. In conditions with low effective intravascular volume having high ADH level, the urea reabsorption can be substantial. Some substances are interfering with the urea estimation ie Falsely high urea level-

aminosaliocylic acid, bilirubin, lipemia, dextran, uric acid, free haemoglobin

Falsely low urea level –

Ascorbic acid, levodopa, and lipids.

2.Serum creatinine:

Serum creatinine is the most widely used indirect measure of GFR. Serum creatinine is very insensitive to even substantial decline in GFR[34]. GFR measured by most accurate techniques may be reduced by up to 50% before serum creatinine becomes elevated. It is insensitive because,

1. Serum creatnine with muscle mass;

Serum creatinine value in young in normal range indicates the normal GFR. The same level in the elder individual could indicate a twofold reduction GFR.[32]

2. Proportional tubular secretion of serum creatinine increases with the decreasing GFR.
3. With declining kidney function extra renal degradation of the creatinine increases.[35,36,37]

Numbers of methods used to measure s.creatinine.

1. Original **Folin – wu** [40]method- using Jaffe reaction.
2. Method of hare – using Lloyd reagent.
3. The direct Alkaline Pictrate method.
4. Auto analyzer using Jaffe reaction.

A number of normal plasma constituents can interfere with creatinine measurement. They are glucose, fructose, pyruvate, acetoacetate, uric acid, ascorbic acid and plasma proteins. These normal constituents cause Jaffe's colorimetric assay to yield high value. These interfering chromogens increase the creatinine value by about 20%.

One study has shown that, in normal individuals the contributions of the nonchromogens in serum creatinine is about 14%, and in CKD patient's nonchromogen contribution in creatinine is about 5%.

Several modifications in Jaffe reaction have done to remove the interfering chromogens. Fuller earth and ion exchange resins used to remove the non-chromogens.

These methods are replaced by less costlier and more convenient auto analyzer technique using the Jaffe reaction. This technique perpetrates the creatinine from non-chromogens. Thus avoids the disadvantage of the standard method.

In our study we utilized the AUTO ANALYSER method [39] using the Jaffe reaction.

3. Creatinine clearence:

The renal clearence of the creatinine is, the urine creatinine excretion divided by the area under the plasma creatinine concentration – time curve for the period in which the urine was sampled.

The creatinine clearance rate more closely resembles the GFR. [28]

The reliability is greatly diminished by

1. Variability of the tubular secretion of creatinine [33]
2. Inability of the most patients to collect the urine properly for 24 hrs
3. Prolonged storage of the urine that falsely raise the urine creatinine by 20%

Creatinine clearance calculated by the Jaffe reaction tends to be falsely low (under estimates GFR). Plasma constituents falsely raise the serum creatinine level, whereas urine creatinine level is largely unaffected. In a given population this error tends to cancel the error introduced by tubular creatinine secretion. The two errors are independent, and the occurrence of the opposing errors of the same magnitude in the same patient is largely a result of chance.

The creatinine clearance is measured by using serum creatinine level and other clinical parameters.

Using the ***cockcroft gault formulae*** the estimated creartinine clearance is measured by this following formulae.[42]

$$\frac{(140\text{-age}) \times \text{weight in kg}}{\text{Serum creatinine} \times 72} \times 0.85 \text{ if female}$$

4. Ultra sound scan abdomen:

This is the most sensitive method to diagnose the chronic kidney disease. In end stage renal disease (ESRD) we expect the kidney size to be reduced and Cortico Medullary differentiation couldn't be differentiated.

The parameters used in ultra sonogram abdomen for the kidney disease are

1. Length – 9 to 12 cms
2. Width - 4 to 6 cms
3. Thickness - <3.5 cms
4. Cortico Medullary Segment differentiation

In chronic kidney disease the size of the kidney is much reduced. The patients is in the need of dialysis usually have the length of the kidney is < 9 cms.

In our study most of the cases have the **kidney size is < 8 cms** in length

Background of the study:

Peritoneal dialysis is an emergency and life saving procedure for the ESRD patients.

In THANJAVUR MEDICAL COLLEGE, we are doing this procedure in NEPHROLOGY unit for the ARF & CRF patients, who were all admitted in the state of pulmonary edema.

Because of the poverty in our area, most of the patients cannot afford for the sophisticated procedure like ***CAPD, CPPD, IPD*** and ***Haemodialysis.*** We are in the need to use the peritoneal dialysis procedure extensively.

References from the journals and Internet, shows lipid profile changes and atherosclerosis events are common in the pts undergoing dialysis. So we conducted this study on the background of these references.

It spite of this procedure being extensively used in this instititution no similar study was conducted previously to evaluate this risk factor, so we conducted this study to evaluate this risk factor and compare it with prevailing trends.

Literature review:

Definition of the chronic kidney disease:

A. Kidney damage for 3 months or longer, as defined by structural or functional abnormalities of the kidney, with or without decreased glomerular filtration rate manifest by either

1. Pathological abnormalities,

2. Markers of kidney damage, including abnormalities in the composition of the blood or urine or abnormalities in the imaging studies.

B. Glomerular filtration rate less than 60ml per minute per 1.73 m^2 for 3 months or longer with or with out kidney damage.[2]

Staging for the CKD

National kidney foundation staging for the chronic kidney disease:

Stage	Description	GFR, ml per m2	Prevalence n (%)	Action

-	At increased risk	>60ml(CKD risk factors)	-	CKD risk reduction
1	Kidney damage with normal or increased GFR	> 90	5,900,000(3.3)	Diagnosis and treatment: treatment of the co morbid condition. Slowing the progression
2	Kidney damage with slightly decreased GFR	60 -89	5.300,000(3)	Estimating the progression
3	Moderately decreased GFR	30- 59	7,600,000(4.3)	Evaluating and treating the complication
4	Severely decreased GFR	15 – 29	400,000(0.2)	Preparation for kidney replacement therapy
5	Kidney failure	< 15	300,000	Kidney replacement

CAUSES OF THE ESRD:

Diabetes mellitus and hypertension are the major causes of the ESRD undergoing the Dialysis.[45]

Glomerulornephritis is the third most common cause of the ESRD undergoing Dialysis.

Other causes for the ESRD:

1. Tubulo interstitial nephritis

2. Infection and reflux nephropathy
3. Polycystic kidney diseases.
4. Haemolytic uraemic syndrome/ thrombotic thrombocytopenic purpura.
5. Vasculitis (wegeners granulomatosis, polyarteritis nodosa)

Risk factors for the progression of the ESRD: [51]

1. Ethnicity
2. Gender – male > female
3. Smoking
4. Heavy consumption of the non- narcotic analgesics.

Treatment modalities for the ESRD: [44]

Haemodialysis

Peritoneal dialysis

Renal transplantation

Types of peritoneal dialysis;

1. CAPD- continuous ambulatory peritoneal dialysis
2. APD- automated peritoneal dialysis
3. IPD – intermittent peritoneal dialysis

Types of haemodialysis :

1. In center haemodialysis
2. Home HD

PRICIPLES OF PERITONEAL DIALYSIS:

Component of the peritoneal dialysis system

1. PD catheter
2. PD solutions
3. Peritoneal membrane and its vascular supply.

I. PD catheter:

Palmer and coworkers first introduced the catheter in 1963 and Tenckoff and Schechter modify it in 1968.

Catheter design:

1. **Acute use catheter** - is straight, relatively rigid conduits about 3 mm in diameter and 25 to 30 mm in length. This catheter is associated with increased risk of infection and malfunction, and this should not be in place for more than 3 days. In ARF pts usually require treatment for longer than 3 days.

2. **Chronic use catheter:**

The standard chronic use catheter is constructed of soft materials like silicone rubber or polyurethane. Silicon catheter is commonly used, and it is biocompatible. Polyurethane catheter has small wall, but cracking of the catheter has been reported.

3. **Catheter design:**

Straight Tenckhoff catheter were associated with high rate of external cuff excursion and migration, so swan neck catheter [46,47] were developed. It has bend and arc to prevent migration and cuff excursion. The downwardly placed exit and placement of the subcutaneous cuffs are designed to decrease the likelihood of the cuff excursion and exit.[48]

4. **Catheter implantation technique**: [50]

General standards of the chronic catheter placement

1. The deep cuff should be in the anterior abdominal wall
2. The subcutaneous cuff should be near the skin surface and not less than 2 cm from the exit site to allow for drainage and provide a firm anchorage.
3. The catheter exit should be placed laterally
4. The catheter exit should be directed downwards and laterally.[57]

5. The intra abdominal portion of the catheter should be placed between the visceral and parietal peritoneum.

The methods used for catheter placement[49] are ***surgical insertion, peritoneoscopic insertion, blind placement, moncrief- popvinch technique***.

II. Dialysis solutions:

Standard dialysis fluid contains electrolytes, buffers, and osmotic agents.

1. Electrolytes:

Sodium: sodium has been added to dialysate fluid in varying range from 120 to140 mEq/l. sodium doesn't cross the peritoneal membrane as readily as the water. The concentrate of sodium in the ultra filtrate is lower than the serum; it is around 70 mEq/l [52,53,54.] · The transient decrease in the dialysate sodium occurs during the dwelling period due to the aquaporin mediated trans cellular water movement that is sodium free, a process called ***sodium seiving***. After multiple exchanges systemic hypernatraemia has been described. To avoid this problem most commercially available dialysis fluids have a sodium concentration of 132mEq/l.

Calcium: Control of calcium and phosphate balance in ESRD is important to prevent the long-term complications of renal osteodystrophy[55,56] . Phosphorous is poorly removed from PD. Because of the complication of the aluminum, used as a phosphate binder, now we are using the calcium in high concentration (3.5mEq/l). This calcium concentration would also correct the hypocalcaemia in an ESRD. This high concentration of calcium would cause hypercalcemia and

metastatic calcification. So in modern ideal dialysis fluid contain the calcium concentration of around 2.5mEq/l. [58,59]

Clinical trials show this calcium concentration would improve the biochemical properties. (s. Calcium, phosphorous, alkaline phosphate, and parathyroid hormone level)

2. Buffers:

Current standard fluid contain lactate and bi-carbonate buffers.

Lactate buffer system: Lactate replaces the acetate[60], which used in the early period as a buffer because of the ultra filtration failure. This filtration failure was due to the development of the sclerosing peritonitis[61]. Acetate buffer would take longer time to reach physiologic PH during the dwell period than lactate will cause increased pain on inflow.

Lactate buffer are available in fluid as D-form and L-form. The important complication of these buffers is they are absorbed and supraphysiologic concentration is associated with encephalopathy.

Bicarbonate buffer system: important disadvantage of this systems are, precipitation of the calcium, magnesium and caramelization of glucose at physiologic PH. To avoid this problems addition of glycyl glycine has been done.[62]

Now the standard buffer system in dialysis fluid is lactate / bicarbonate mixture.

4.Osmotic agents:

Glucose: it is safe, effective, readily metabolized and inexpensive. Glucose is not an ideal osmotic agent because,

i. Rapid absorption

ii. Potential for metabolic complications, hyperglycemia, hyperinsulinemia, hyperlipidemia, obesity.[23,24]

iii. Necessity for an acidic dialysis PH to prevent caramelization.

iv. Potential non-enzymatic glycosylation of peritoneal tissue during periods of mesothelial cell loss.

Amino acids: protein malnutrition is a significant risk for morbidity and mortality in dialysis patients. Because of the dialysis loss of protein and amino acids into the PD, amino acids containing fluid would be the caloric source. Other benefits of amino acid absorption is, that to cause intra peritoneal vasodilatation, that increasing the surface area for the dialysis[63]. The 1.1% amino acids effectively replace the protein loss through the dialysis fluid.

The main disadvantage of the amino acids is the development of the metabolic complications and increased levels of serum urea nitrogen level

Polypeptides and oligopeptides :

Main advantages of polymers are

1. Prolonged ultra filtration, because of higher average molecular weight
2. Presence of ionized branched chain that provide higher osmotic pressure on a molar basis,
3. Potential for providing protein based calories.
4. It lowers serum cholesterol level and reduces serum insulin level and insulin sensitivity.

Main disadvantage of polymers; Rash, Exfoliative reactions, Sterile peritonitis

Advantage of poly glucose over glucose a comparison;

1. Glucose induces trans capillary ultra filtration across both small inter endothelial and ultra small trans cellular pores; whereas poly glucose induce ultra filtration across inter endothelial pores.
2. Glucose and poly glucose have markedly different trans capillary ultra filtration profiles.
3. Ultra filtration with glucose is rapid and occurs in early period and decreases with time; whereas poly glucose ultrafiltration increases linearly with time.
4. Glucose is absorbed so that crystalloid induced osmotic gradient is no longer present, ultra filtration ceases and lymphatic absorption of fluid predominates; whereas poly glucose is slowly absorbed through lymphatics.[64]

An ideal peritoneal dialysis solution: [65]

1. Good solute clearance and ultra filtration capacity
3. Necessary solutes supplied and uremic toxins removed.
4. Nutrition supplied or does not promote catabolism.
5. Isosmolar solution, normal pH, bicarbonate as buffer
6. Minimal absorption of osmotic agent
7. Anti bacterial and anti fungal properties.
8. Membrane biocompatible/ doesn’t promote chronic inflammation.

III. Peritoneal membrane:

Anatomy;

Membrane is the primary interface between the blood and the Dialysate fluid. It contains two principal parts

1. Parietal peritoneum – 10% of the total
2. Visceral peritoneum - 90% of the total

The Total surface are of the Peritoneal membrane is 1 to2 m^2. Children have disproportionately larger peritoneal surface than adults. The Peritoneal surface is continuous and closed in males. In females it is continuous with the mucous membrane of the fallopian tube. Intra abdominal opening is normally collapsed; there is no free communication. The Peritoneal cavity contains about 100ml or less of fluid normally. It has the capacity to accommodate 2 liters or more.

Mesothelium:

This is a continuous monolayer of flattened cells, which about 0.5 mm thick. The free surface of the Mesothelium is covered by microvillus that increases the surface area. It contains tight junctions, which have an anchoring between cells of one another and basement membrane [66,67]. It contains gap junctions, which mediate the passage of chemical or electrical signals. Mesothelial cells are ultra structurally similar to Type II Pneumocytes [68]. They contain the lamellar bodies identical to those Pneumocytes. These cells secrete surfactant like lubricant material.

Sub Diaphragmatic stoma, allows the direct contact between the peritoneal fluid and lymphatic, through which the peritoneal fluid got absorbed.

Basement membrane:

It is a homogenous membrane contains openings in the diaphragmatic surface, made up of Type IV collagen proteoglycans and glycoproteins. It is a selective barrier by preventing the fibroblast from contacting Mesothelial cells.

Interstitium:

It is a primary support for the Peritoneum. It is made up of the Mucopolyschariде matrix. The Interstitium contains the aqueous and lipophilic phases for the transport of the substances.

Blood vessels:

The celiac and mesenteric arteries supply visceral peritoneal membrane with venous drainage via portal veins.

Parietal peritoneum is supplied by circumflex, iliac, lumbar, inter coastal and epigastric arteries with drainage into the systemic veins.

The capillary wall contains at least two different pores, larger pores situated in the Venular end and the small pores situated in the capillary end.

Peritoneal lymphatics:

Intersitial fluids and solutes are removed by the lymphatic vesssels. They also function to maintain 50 to100ml of fluid in the Peritoneum. The fluid absorption mainly occurs through the stomas in the sub-diaphragmatic peritoneum.

PERITONEUM AS A DIALYSIS SYSTEM:

There are potential resistance sites present in the peritoneal membrane to transport the fluid and solutes. They are

1. Fluid films within the capillary lumen
2. The endothelial layer
3. The capillary basement membrane
4. The Interstitium
5. The Mesothelial layer
6. Fluid film in the peritoneal cavity.

The solute transport occurs by both diffusive [70] and convective forces. The mass transport barrier appears to offer very little resistant to diffusion process, and offer significant resistance to the convection process. Resistance to flow of solute is greater than resistance to flow of water.

Intracapillary fluid films - exert little resistance for the low- molecular-weight solutes [69].

Capillary endothelium – It is a selective barrier to solute transport, low resistance barrier to low molecular weight solutes, and offer significant resistance to molecular, which increases further. The transport across the capillary endothelium is defined as a three-pole model.

1. Water channels – analogous to aquaporins, these channels are sensitive to crystalloid-driven osmosis.

2. Small pores – this inter endothelial clefts with a radius of 4 to 6 nm – 90 to 93% of the total pole area. These poles restrict the passage of the proteins, but allow the urea, creatinine and water.
3. Large pores – which have radii larger than 20 nm and are probably located in the venular side of the pore area [71].

Diffusion is postulated to occur through large pores located at the Venular end of the capillaries.

Convection occurs through the small poles at the arterial end of the capillaries.

Capillary basement membrane:

This membrane produces little effect on the diffusion of low molecular – weight solutes. The anionic sites predominantly composed of Heparin sulphate and Chondroitin sulphate, inhibit the transport of the charged solutes.

Interstitium:

The longest distance the solute must traverse. This is the major site of resistance for urea and low molecular weight solute. The gelatinous mucopolyscharide matrix interspersed with water rich, colloid poor membrane offers the resistance. The fixed ionic charges on the collagen and mucopolysacharides may influence the transport of charged solutes.

Mesothelium:

This is more permeable than endothelium; because of the larger inter cellular gaps. Visceral mesothelium is more permeable. Surface-active phospholipids adsorbed on to the mesothelial lining and maintaining the integrity of the semi-permeable properties of the membrane.

Complication of the peritoneal dialysis:

1. Cardiovascular complications
2. Infectious complications
3. Non infectious complications

1. Cardiovascular complications:

Mortality and morbidity from cardiovascular disease are greatly increased in patients on maintenance dialysis therapy. Cardiovascular mortality continues to account for more than 50% of death in haemodialysis patients. The cardiac mortality can arise from arrythmia, cardiomyopathy, and ischemic heart disease.

The first report of accelerated atherosclerosis in dialysis patients originated in the 1970s from scribe and colleagues based on their experience. Their report with ACSVD prevalence varies from 24% in non-diabetic patients on dialysis to as high as 85% of diabetics on dialysis. Their post morterm examination report of coronary atherosclerotic disease in dialysis and control confirm more calcified plague in patients with ESRD on dialysis. Increased medial thickness is characteristic of coronary plagues in dialysis.

Hypertension:

This is very common and occurs in 50 to 90 % of the PD patients. Hypertension in PD patients is due to fluid retention as a result of the impaired ultra filtration [72]. This increases the risk of the LVH and cardio vascular morbidity and mortality. Prescribing the loop diuretics, preservation of the residual renal function, reduction of dietary salt intake and limitation of fluid intake and prevention and treatment of the ultra filtration failure can prevent this complication.

Hyperlipidaemia:

PD is associated with increased glucose load because of the constant absorption from the peritoneal cavity [23,24]. So the patients have constant susceptibility to the development of the hyperinsulinemic state. This hyper insulinemic state increases the synthesis of triglycerides in the liver [8]. The dialysis procedure results in loss of 5 to 15g of protein per day, preferably small molecular weight proteins, so there is a loss of HDL lipoprotein [73] is the rule.

Initiation of the PD significantly increases the synthesis of serum cholesterol, serum triglycerides and total cholesterol [3,4,6,7] levels. The total cholesterol shows the predominant increased in the LDL level. The strongest predictors of the lipid profiles are weight gain, higher plasma glucose level and preexisting cardiovascular co morbidity. The serum lipid protein analysis shows elevated levels of Lp (a) [5] and apo proteins, including apo-A and apoB containing lipoproteins, apoCIII, apoB, apoC levels. The increased Lp (a) level is due to increased synthesis, not due to decreased clearance. Lp(a), an atheorogenic lipoprotein consists of low density lipoprotein cholesterol particle that is covalently bonded to apolipoprotein (a), a glycoprotein with genetic polymorphism.

An inception cohort study of incident dialysis patients followed prospectively demonstrated that small apo lipoprotein (a) size predicts mortality, even with multiple adjustments for demography, comorbidity, cause of renal failure and congestive cardiac failure.

The dietary modification shows minimal reduction in the serum lipid levels. The use of 1.1% amino acid dialysate exchange per day did not report any improvement. The use of polyglucose solutions has been associated with a decrease in lipid levels [22].

Hypercholesterolaemia in PD patients has been successfully treated with atorvastatin and gemfibrosil. Hyper triglyceridemia has been treated with atorvostatin, fibric acid derivatives and fish oil supplementation.

C- reactive protein:

PD patients have serologic evidence of an enhanced inflammatory response [10,11]. Factors that cause the enhanced inflammatory response include the decreased renal clearance, the biocompatible dialysate solutions and co morbid medical conditions. The acute phase reactants such as CRP, Lp(a), Fibrinogen and proinflammatory cytokines(IL 6) are elevated, they have direct atherogenic properties [14,15,16]. Plasma levels of C-reactive protein as a marker of acute inflammatory reaction have been demonstrated in prospective cross section studies and in cohort studies to be a powerful predictor of cardio vascular disease and all cause mortality in dialysis patients. Approximately 30 to 60 % of PD patients have elevated CRP levels.

Hypoalbuminemia is also associated with elevated CRP level. It is a negative acute phase reactant. The strong association between atherosclerosis, elevated CRP, and hypoalbuminemia

has lead to the term Malnutrition Inflammation Atherosclerosis syndrome (MIA syndrome) to describe the micro inflammatory process.

The presence of the pro inflammatory cytokines IL-6 has similarly been associated with the presence and subsequent progress of underlying atherosclerosis in dialysis patients.

Homocysteine:

Most PD and HD patients have elevated homocysteine levels; patients with low folate or vitamin b12 levels are at increased risk of having elevated homocysteine [16,17,18,19] levels. PD removes Homocysteine; the removal is not high enough to allow for the correction of the homocysteniemia. The amino acid containing PD solutions, which have methionine, cause an increase in homocysteine level. Elevated homocyteine in long-term dialysis patients predicts the ASCVD.

Oxidative stress:

Oxidative stress is also highly prevalent in haemodilysis patients and may contribute to an acceleration of atheroscelrosis. Oxidation of LDL, particularly in the subendolthelial space, leads to uptake of oxidized LDL by monocyte- macrophage, and conversion in foam cells, the earliest stage in atherosclerotic process. Oxidative stress may occur in dialysis patients directly as a result of impaired renal clearance of oxidants and by increased production by activated macrophages. This increased oxidative stress contributes to cardio vascular disease, evaluated in SPACE study. (Secondary prevention with antioxidants of cardio vascular disease in end stage renal disease)

Other cardio vascular risk factors:

- Nocturnal hypoxemia may also be an important predictor.
- Corrected QT dispersion (difference between the shortest and longest QT intervals) is a meaure of regional heterogenicity in myocardial repolarization.
- Asymmetrical di-methyl arginine, an endogenous nitric oxide inhibitor that accumulates in uraemia, shows significant correlation with atherosclerosis.

Angina in dialysis patients:

The presence of severe coronary artery disease without symptomatology is commom in HD patients.

75% of diabetic patients with angiographically significant CAD had no symptoms. Similarly findings have also been identified in non-diabetic HD patients. The lack of anginal symptoms in non-diabetics may be explained by uraemic autonomic neuropathy or by low level of physical activity in long-term dialysis patients.

To evaluate the asymptomatic CAD, Ambulatory electro cardiographic recordings have not been well studied in HD patients. Exercise electrocardiography is affected by abnormal resting electrocardigram and by markedly reduced exercise tolerance. There are conflicting reports about usefulness of Stress Echocardiography and Exercise stress test. Relatively high sensitivity and specificity has been reported for Dipyridamol exercise Thallium Imaging and Dobutamine stress Echocardiography.

II. Non- infectious complications:

Mechanical complications;

In flow pain

Outflow failure

Catheter damage

Intra abdominal pressure:

Hernias

Dialysate fluid leak

Hydrothorax

Alterations in the respiratory function

Back pain

OBSERVATION NO 1

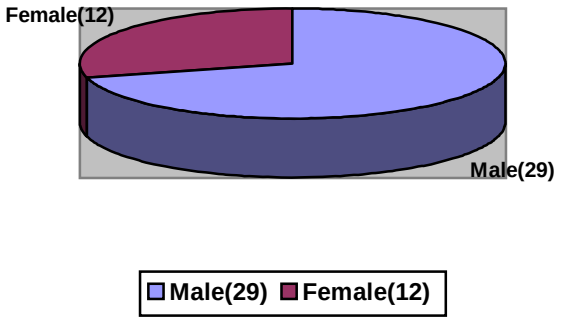
General character of the study group;

41 patients were included in this study, among them 29 were male and 12 were female. All of them were known hypertensive patients. The duration of the hypertension varies from 1½ yrs to 15 yrs.

When analyzing the body weight of the study population, their body weight ranges from 38 kgs to 70 kgs. Mean body weight of the male patients was 63.13 kgs. Mean body weight of the female patients was 55.1 kgs

All the patients involved in our study were non-diabetics. Their mean blood sugar levels were 101 mgs%.

All the 41 patients satisfied the criteria for the peritoneal dialysis. Their mean urea levels were 148.5 mgs% and mean creatinine levels were 11.09 mgs%.

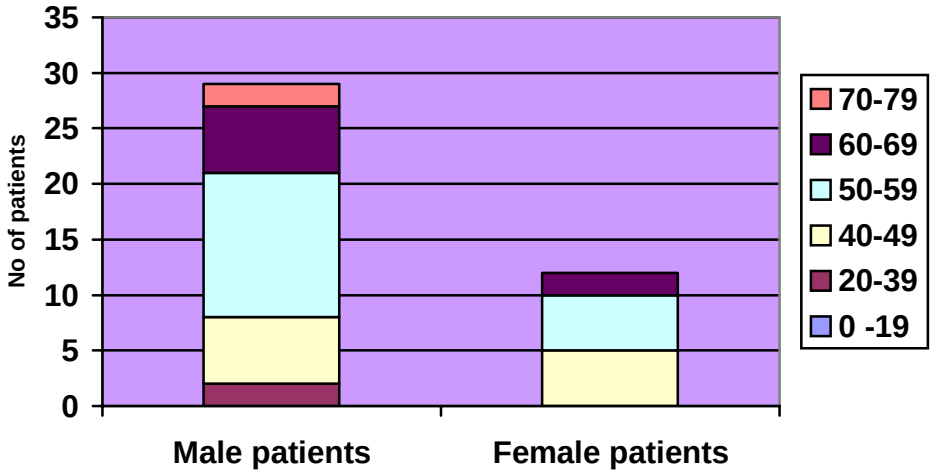


OBSERVATION 2:

Analysis of age distribution in the study population;

Age group	Male patients	Female patients	Total no of patients
0 -19	0	0	0
20-39	2	0	2
40-49	6	5	11
50-59	13	5	18
60-69	6	2	8
70-79	2	0	2
Total	29	12	41

The age group of our study population falls between 38 –70 years of age. Most of the study population falls between the ages of 50 –59 yrs in our study. The mean age of the study population is 52.5 yrs.



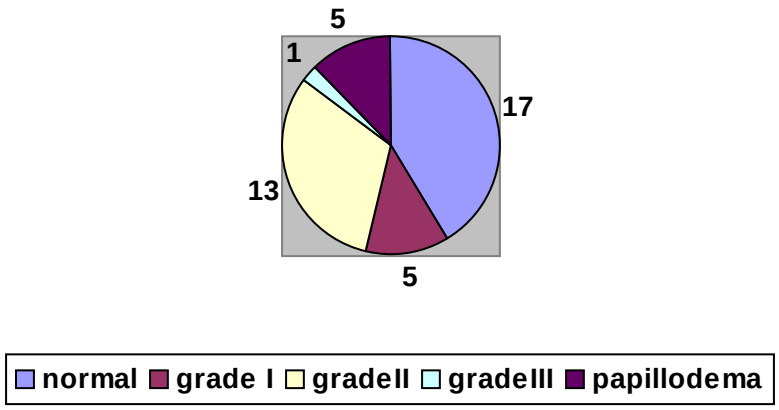
OBSERVATION 3

ANALYSIS OF THE PATIENTS WITH HYPERTENSIVE RETINOPATHY:
Total no. of patients with retinopathy –24.

Fundus changes	No of patients
Normal	17
Grade I	5

Grade II	13
Grade III	1
Pappiloedema	5

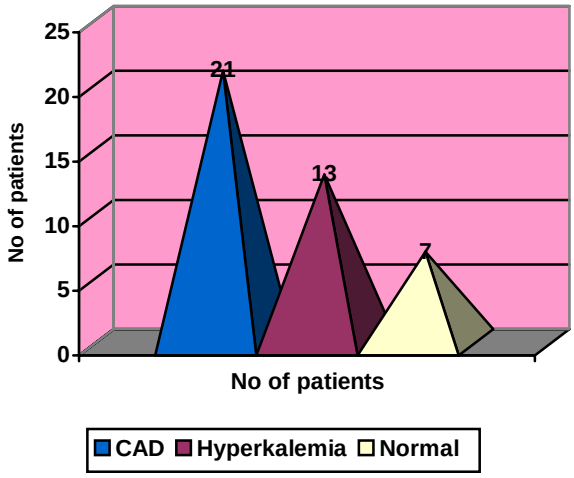
Opthalmological examination revealed the fundoscopic changes in our study group. 59% of male patients (17 males) and 58% female patients (7 females) have hypertensive retinopathic changes. Among the patients, 17 have normal fundus, 13 have grade II retinopathy changes, 5 have pappilloedema, 5 have grade I changes and 1 patient have grade III changes



OBSERVATION 4

ECG features	No of patients
CAD	21
Hyperkalemia	13
Normal	7

When analyzing the electrocardiographic finding of the study group, 21patients (51.2%) had the features of coronary artery heart disease, 13 patients (31.7%) had features of hyperkalemia and 7 patients (17%) had normal findings. The CAHD patients were not on anti ischemic therapy previously

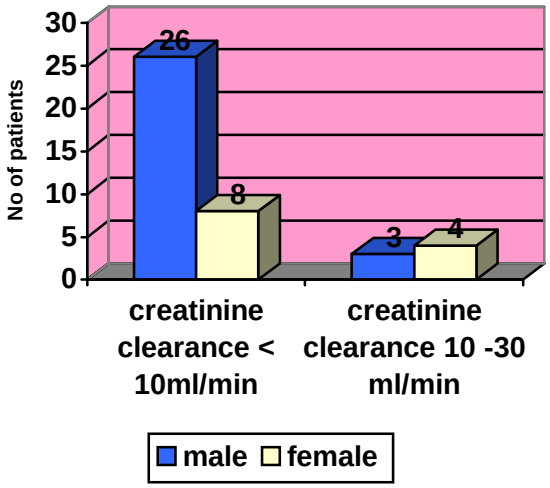


OBSERVATION 5

Analysis of the creatinine clearance of the study population

	Male	Female
Creatinine clearance <10 ml/min	26	8
Creatinine clearance 10-30ml/min	3	4

Among the 41 patients in our study, most of the patients have their creatinine clearance of < 10 ml/min. 90% of the males (26 patients) and 67% of females (7patients) had their creatinine clearance < 10 ml/min. Rest of the patients had their creatinine clearance falls between 10 – 30 ml/min. their mean creatinine clearance was 7.4 ml/min.



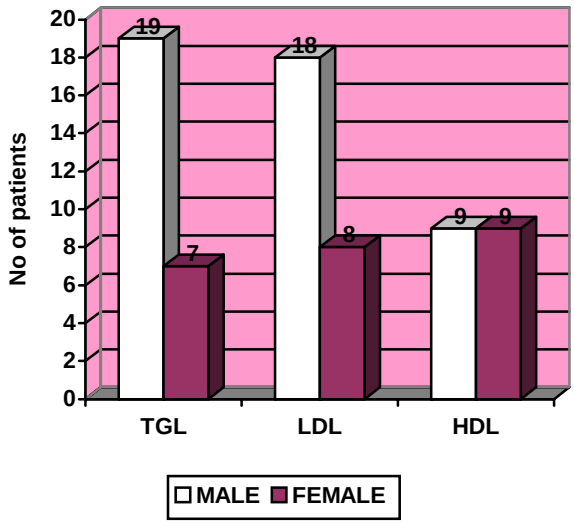
RESULT 1

Patients with post dialysis elevation of ATHEROGENIC LIPIDS in the serum (expressed in no.)

s.no		Male	Female
1.	Increase in TGL	19	7
2.	Increase in LDL	18	8
3.	Reduction in HDL	9	9

After 48 hrs of continuous peritoneal dialysis 19 out of 29 males (65%) and 7 out of 12 females (58%) had the rise in Triglyceride levels, 18 out of 29 males (62%) and 8 out of 12 females

(67.5%) had rise in the low density lipoprotein levels and finally 9 out of 29 males (31%) and 9 out of 12 females (78%) females had a reduction in the High density lipoprotein levels

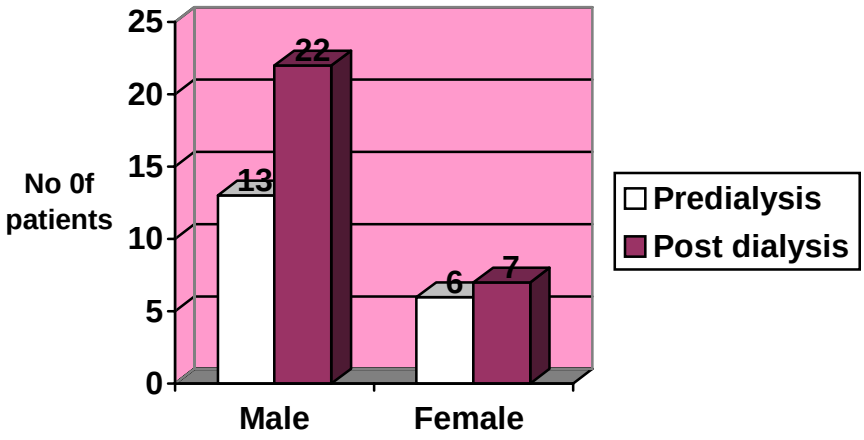


RESULT 2

Analysis of the number of patients with elevated TGL level in the serum

	Male	Female
Predialysis	13	6
Post dialysis	22	7

In the study population, 13 male patients and 6 female patients has rise in their serum TGL level above 150 mgs/dl. Most of them show borderline elevation around 150% mgs to 160 mgs%. In the post dialysis sample, 22 males and 7 females has significant rise in the serum TGL level. Most of the serum TGL levels exceeds 180 mgs%. This rise is very significant.

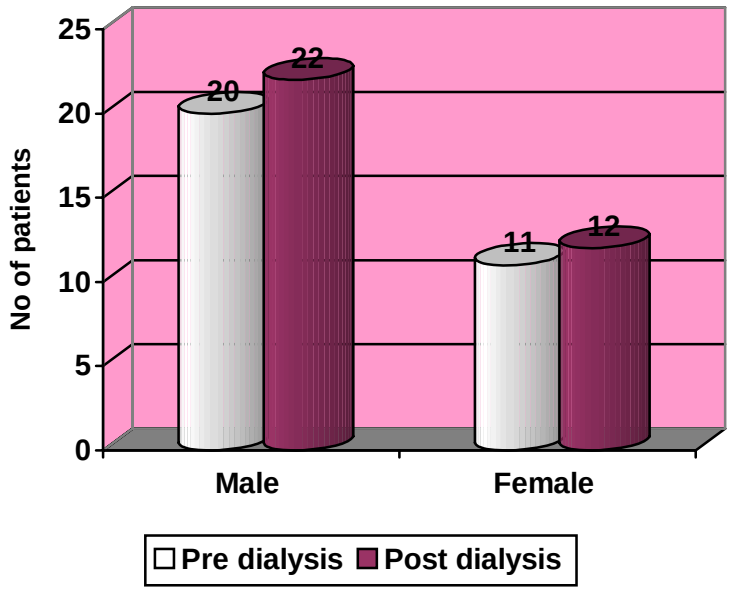


RESULT 3

Analysis of the patients with elevated LDL level in the study group.

	Male	Female
Pre dialysis	20	11
Post dialysis	22	12

When observing the pre dialysis LDL level, 20 out of 29 males and 11 out of 12 females had elevated LDL in their serum. In post dialysis sample 22 males and 12 females has elevated LDL level in their serum.

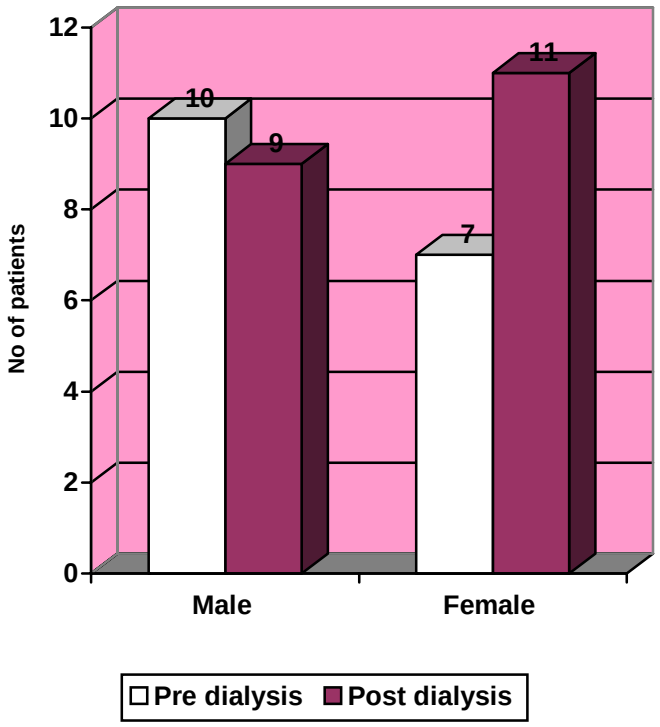


RESULT 4

Analysis of the patients with reduced HDL level in the study population

	Male	Female
Pre dialysis	10	7
Post dialysis	9	11

By observing this table the number of patients with HDL fall is more in the females than males.



RESULT 5

Analysis of the mean TGL in the study population

Pre dialysis mean TGL

Male- 4418/29 = 152.34 mg/dl

Female –1873/12 = 156.08 mg/dl

Post dialysis means TGL

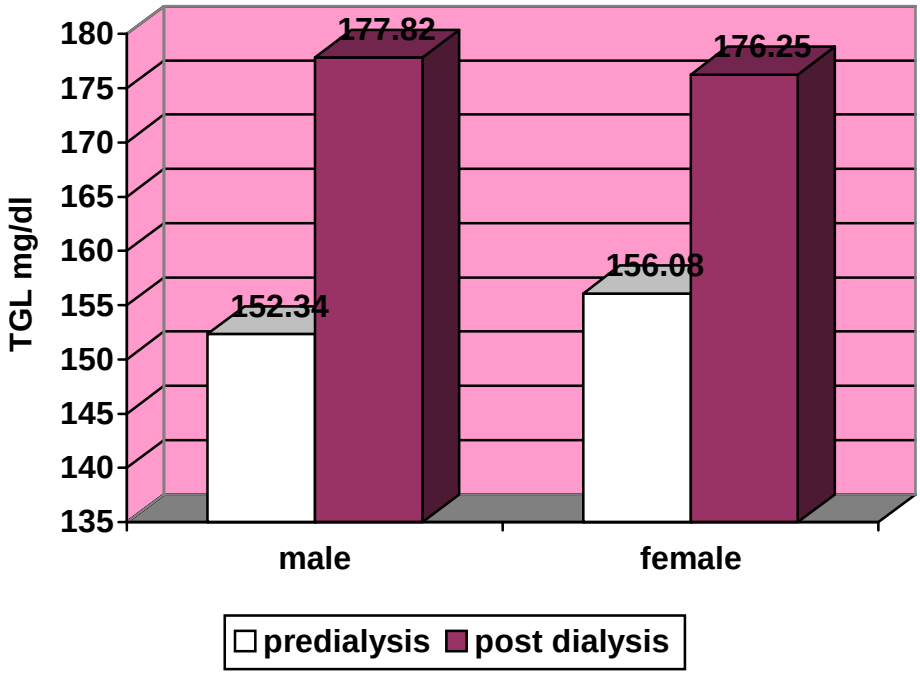
Male – 5157/29 =177.82 mg/dl

Female - 2115/12 =176.25 mg/dl

	Male	Female
Pre dialysis	152.34	156.08
Post dialysis	177.82	176.25
P value	<0.05	>0.05

The mean pre dialysis TGL level in males and females are 152.34 mgs /dl and 156.08 mgs /dl respectively. This level rose to 177.82 mgs /dl in males and 176.25 mgs /dl females after the dialysis. P value shows the 5% level significant difference between two means in males, but insignificant difference of 5% level between two means in case of females.

Analysis of mean TGL in study population



RESULT 6

Analysis of the mean LDL level in the study group:

Pre dialysis mean LDL

Male –3937/29 =136.31 mg/dl

Female –1712/12 =142.67 mg/dl

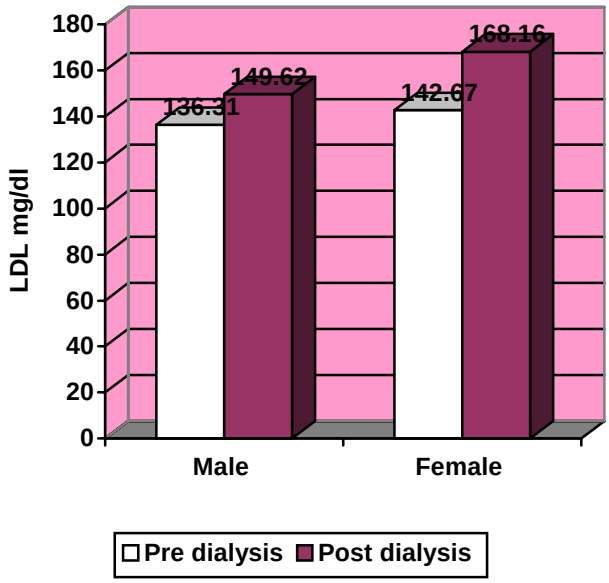
Post dialysis mean LDL

Male –4340/29 =149.62 mg/dl

Female –2017 /12 =168.16 mg/dl

	Male	Female
Pre dialysis	136.31	142.67
Post dialysis	149.62	168.16
P value	>0.05	>0.05

P value shows statistically insignificant difference of 5% level between the two values in males and females.



RESULT 7

Analysis of the mean HDL level in the study

Pre dialysis mean HDL

Male – 1136/29 = 44.31 mg/dl

Female –570/12 = 47.5 mg/dl

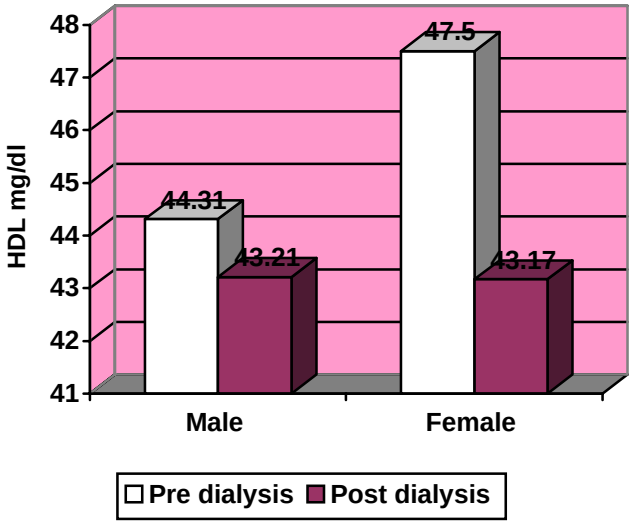
Post dialysis mean HDL

Male – 1233/29 = 43.21 mg/dl

Female – 578/12 =43.17 mg/dl

	Male	Female
Pre dialysis	44.31	47.5
Post dialysis	43.21	43.17
P value	>0.05	>0.05

P value shows statistically insignificant 5% level between the two means in case of males and females.



Discussion:

According to the ATP III guidelines:

Risk factors for the atherosclerosis are:

1. Central obesity (waist circumference) - >102 cms (males), >88cms (females)
2. Hypertriglyceridemia ->150 mg/dl
3. Low HDL - <40 mg/dl (males), <50 mg/dl (females)

4. Hypertension >135/85 mmHg

5. Fasting blood sugar 110 –125 mgs%.

The atherogenic lipoproteins in the blood in patients undergoing peritoneal dialysis are elevated LDL, elevated Triglycerides, elevated Lp (a) level and low HDL. According to the ATP III 2001 guidelines the dialysis is an atherogenic event.

The apoproteins present predominantly in the LDL and TGL are apoB 100. The apoprotein present predominantly in the HDL are apoA-I, which is protective to the atherogenic event.

The apoB48, present predominantly in the chylomicrons are not much atherogenic.

LDL is the cholesterol contains much rise in the apoB100 level, which is atherogenic. When this LDL is associated with the high TGL level the incidence of the atherogenisis is significantly elevated. Multiple lipid abnormalities are more atherogenic.

Low levels of the HDL are an indirect evidence of the elevated apoB100 level.

In the studies we had taken for the references most of them dialysis were done for prolonged period. The study conducted in *Sweden, sahlgreska university hospital shows* [12;] their study period was 6 to 48 months. Their mean study period was 15.3 months. They had utilized the intermittent peritoneal dialysis procedure. They had monitored the bi-weekly creatinine clearance and glucose level in the study population. They had done the peritoneal dialysis procedure for the non-diabetic patients. In their study, they had significant rise in total cholesterol (7.1 mmol/ l), VLDL –cholesterol (1.0mmol/l), LDL cholesterol (4.7 mmol/l) and triglyceride level (2.5mmol/l). They had statistically analyzed their results showed significant

difference present between the analyses samples. They had analyzed in 95% and 99% confidence levels.

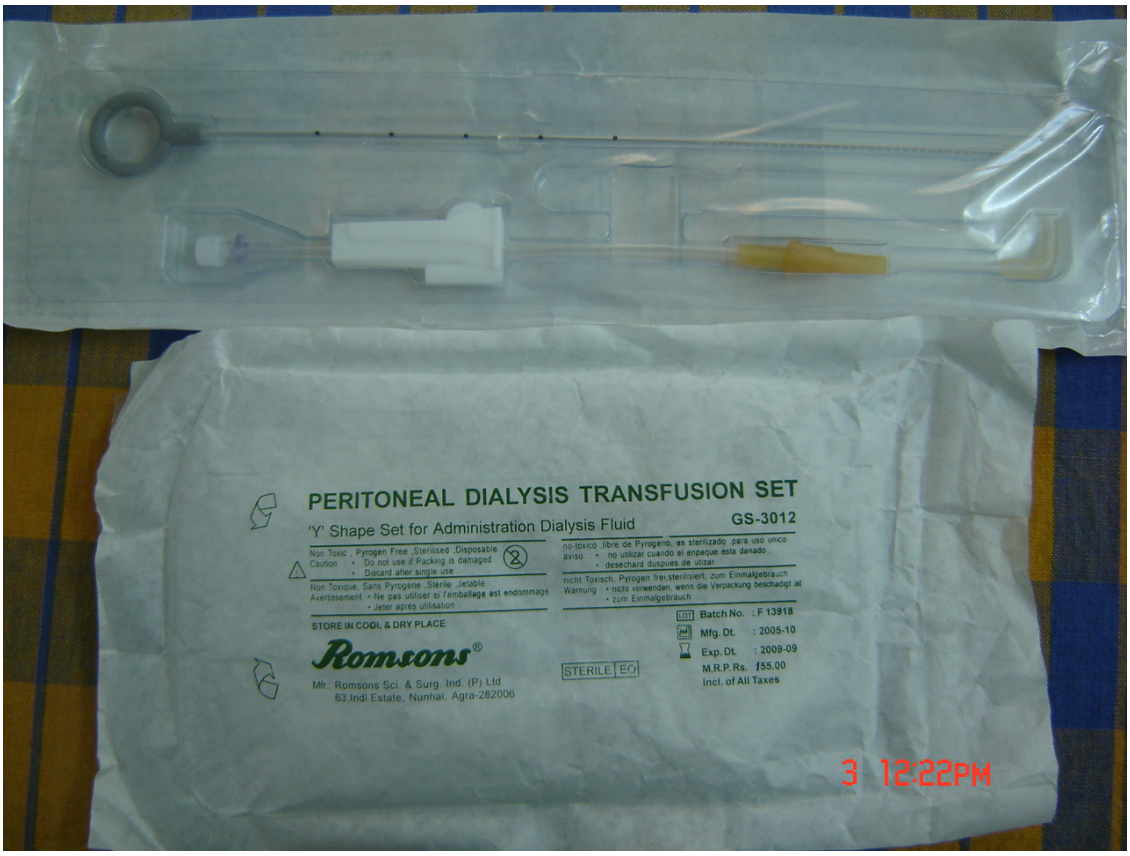
We have taken the ATP III guidelines and the above standard study as our reference we had undergone the study and analyzed the results.

We had taken the study population from the low socioeconomic status group. After satisfying the inclusion and exclusion criteria we had done the peritoneal dialysis for continuous 48 hr period, whereas in the standard reference study, the study period varies from 6 months to 48 months. In spite of these short 48 hrs period analyses, there is a rise in serum triglycerides (177.2mg/dl), serum LDL (168.08mg/dl) and serum HDL (43.1 mg/dl) were observed in our study. But when we statistically analyzed the results with *paired t test* in 95% confidence level, the rise in triglyceride in male patients is significant at 5% level. The rise in LDL and reduction in the HDL are statistically insignificant at 5% level.

Even though there is a rise in serum lipid levels in our study, the statistical analysis reveals the rise is insignificant at 5% level. When we compare to the reference studies, the duration of dialysis in our study is minimal (48 hrs). When we do the dialysis for longer period and by increasing the sample size, we can make our study statistically significant.

Conclusion:

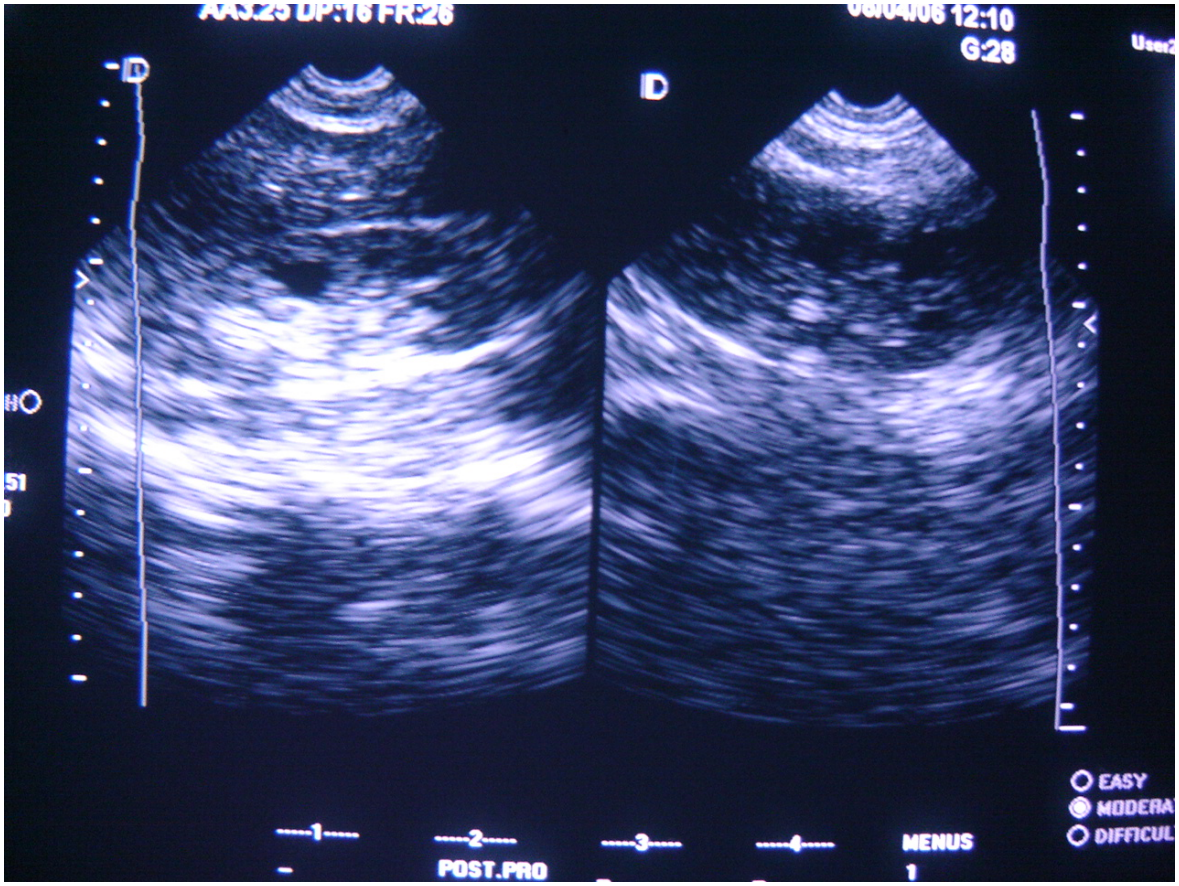
1. By doing the peritoneal dialysis in the ESRD, we should expect the elevation of the serum TGL, LDL & reduction HDL levels.
2. By doing the peritoneal dialysis the Triglycerides in males and low-density lipoproteins in females are significantly elevated.
3. The lipid elevation in peritoneal dialysis reaches the atherogenic level, according to the ATP III criteria; so peritoneal dialysis is also the one of the cardiovascular risk factor in ESRD patients.
4. Anti Dyslipidemic agents like Stains and Fibrates might be prescribed in the patients undergoing long term peritoneal dialysis.



Picture 1: Shows the material for peritoneal dialysis; the catheter and the infusion set.

Picture 2: The peritoneal dialysis on progress. Properly positioned catheter and its haemostasis.

Picture 3: The peritoneal dialysis on progress, in a 70 yrs old man with chronic kidney disease.



Picture 4: Ultrasonogram features contracted kidney. Size RK –8.6X3.6, LK – 8.7X3.4

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MASE

					BASIC DISEASES		
S.NO	NAME	AGE	SEX	IP.NO	DM	HT	CAHD
1	Mr.RAMADOSS	40	male	856328	nil	3 yrs	nil
2	Mr.RAVIKUMAR	40	male	855688	nil	7yrs	yes
3	Mr.ARUMUGAM	60	male	856383	nil	7 yrs	IWMI
4	MR. KRISHNAMORTHY	60	male	850928	nil	15yrs	nil
5	Mr.RATHNAVEL	65	male	846027	nil	5 yrs	nil
6	Mr.NAVANEETHAN	55	male	840618	nil	2 yrs	nil
7	Mr. SADASIVAM	40	male	849668	nil	3 yrs	nil
8	Mr. NAGARAJAN	70	male	850783	nil	7yrs	nil
9	Mr. MOHAN	48	male	852122	nil	9 yrs	nil
10	Mr.PERUMAL	54	male	843424	nil	3 yrs	nil
11	Mr. SUSAI	50	male	839308	nil	3 yrs	nil
12	Mr.SESIAPPAN	59	male	852960	nil	7yrs	nil
13	Mr.Durai arumugam	70	male	839180	nil	12yrs	nil
14	Mr.AROCKIASWAMY	54	male	845217	nil	9yrs	nil
15	Mr.THANGAVEL	65	male	845212	nil	6yrs	nil
16	Mr. MOHAMMED	54	male	853337	nil	11yrs	nil
17	Mr.KANNAN	65	male	848133	nil	11yrs	nil
18	Mr. SHANKAR	50	male	852816	nil	7yrs	nil
19	Mr.SELVRAJ	45	male	842290	nil	1i/2 yrs	nil
20	Mr.MURUGAVEL	56	male	851666	nil	5yrs	nil
21	Mr.RAJENDERAN	38	male	843548	nil	3yrs	nil
22	Mr. RAJARAM	50	male	852671	nil	2yrs	nil
23	Mr.KULANDAISAMY	55	male	847291	nil	11/2yrs	yes
24	Mr.ARIVALAGAN	45	male	848289	nil	4yrs	nil
25	Mr.RENGASWAMY	60	male	859991	nil	7yrs	yes
26	Mr.KUMAR	38	male	851626	nil	3yrs	nil
27	Mr.PACKRISWAMY	55	male	841602	nil	4yrs	nil
28	Mr.KUMAR	40	male	856755	nil	2yrs	nil
29	Mr.SANGAMUTHU	50	male	847646	nil	2yrs	yes
30	Mrs.SELVI	48	female	847885	nil	2yrs	nil
31	Mrs.RENUKA	45	female	844338	nil	5yrs	nil
32	Mrs.SANKARAMMAL	54	female	853816	nil	4yrs	nil
33	Mrs.VIJAYA	53	female	853291	nil	7yrs	nil
34	Mrs.ANJALI DEVI	40	female	849499	nil	7yrs	nil
35	Mrs. VIJAYALAKSHM	63	female	853241	nil	11yrs	nil
36	Mrs.KURUVAMAL	65	female	843417	nil	15yrs	yes
37	Mrs.DHANALAKSHMI	45	female	860344	nil	6yrs	yes
38	Mrs. LAKSHMI	58	female	851141	nil	3yrs	yes
39	Mrs.AKILAMBAL	40	female	843275	nil	8yrs	yes
40	Mrs.KAMALA	56	female	849537	nil	11yrs	yes
41	Mrs.VINNARASI	54	female	844967	nil	6month	nil

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ETR CHART CONTAIN THE DATAS FOR THE DYSLIPIDEMIC CHANGES IN PATIENTS UNDERGOI

WEIGHT	BP	FUNDUS	SUGAR	UREA	CREATININE	SODIUM	S. K	CR.CL
kgs	mm/Hg		mgs%	mgs%	mgs%	mEq/l	mEq/l	ml/min
60 kgs	170/110	grade II	68	120	7.9	119	5.4	10.5
53	160/100	grade I	106	229	20.1	132	3.8	3.6
70	180/110	grade I	109	203	10.1	132	4.2	7.7
52	170/110	grade I	80	117	12.7	130	12.7	4.5
70	180/110	normal	118	186	10.8	132	5.3	7
63	160/110	papiloedema	81	78	5.2	135	5.6	11.4
69	160/110	normal	95	134	7.7	116	5.9	12.4
63	160/110	grade I	98	133	5.5	128	5.9	12
57	180/110	normal	111	140	11.2	134	5.2	6.5
51	180/110	grade II	106	160	9.9	120	6.8	6.1
52	190/110	grade II	98	164	17.2	132	6.1	3
70	200/130	papiloedema	82	127	10.5	136	5.2	9
63	140/100	grade III	140	120	7.5	139	5.3	8.2
58	150/110	grade II	107	105	10.2	130	5.6	7.5
59	160/110	grade II	96	133	12.8	128	6.2	4.8
57	17/110	grade II	98	164	16.4	126	5.7	4.2
68	180/90	gradeII	113	134	13.3	127	3.4	5.3
68	200/120	normal	90	180	8.3	131	5.8	10.2
70	160/100	normal	82	127	10.5	136	5.2	9
70	160/100	normal	130	190	10.2	130	5.6	8.5
59	130/100	normal	96	140	10.9	128	5.6	7.6
70	180/90	normal	146	190	10.2	130	5.6	8.5
63	170/100	normal	82	165	10.8	117	6.4	5.5
59	140/100	normal	74	148	10.6	119	6.8	8.7
73	170/100	normal	88	235	12.5	128	6.8	6.5
58	180/100	normal	84	144	11.6	128	5.9	7
67	190/120	grade II	96	157	9.3	130	4.8	6
53	180/100	normal	96	240	18.2	133	5.1	4
48	210/120	papiloedema	130	145	13.5	126	7.3	5.8
38	140/100	grade II	130	105	10	123	5.4	5.9
55	160/100	normal	107	188	22.2	123	6.6	3
63	170/100	grade II	120	130	8.6	130	3.7	9
46	230/130	papiloedema	85	108	6.7	134	5.1	11.1
41	180/120	grade II	136	149	15.1	128	6.1	4
56	230/130	papiloedema	85	108	6.7	134	5.1	11.1
61	160/110	grade II	70	120	10	137	5.8	5.4
63	170/120	grade I	106	138	7.8	130	5.6	11
59	150/110	normal	98	94	5.6	132	5.6	10.1
65	170/100	normal	79	172	20.2	116	9.8	4
68	150/90	normal	126	173	5.1	133	5	15.5
46	160/110	normal	106	92	11.3	124	4.2	4.1

NG PERITONEAL DIALYSIS

			S. LIPID PROFILE - mgs%						
USG. ABD		ECG	PRE DIALYSIS				POST DIALYSIS		
RK- cms	LK- cms		TGL	LDL	CHOLESTROL	HDL	TGL	LDL	CHOLESTROL
8.1x3.3	7.8x3.1	normal	136	136	190	53	138	103	179
7.2x3.9	7.3x3.3	HT	86	148	203	38	70	103	194
8.8x4.4	7.1x4.3	CAHD	176	93	180	52	161	94	176
7.3x3.8	7.4x3.2	IWI	146	87	149	33	119	92	145
8.1x 3.6	8.3x4.5	tall T	52	140	224	54	160	110	200
7.4x3.1	7.4x3.0	tall T	240	176	272	50	272	196	298
7.2x4.2	7.5x3.8	IWMI	237	159	243	36	309	131	238
7.3x4.5	7.9x3.6	LWI	240	176	272	50	272	196	298
9.5x4.1	8.3x4.2	normal	135	108	170	35	165	67	149
8.0x4.1	7.6x4.2	tall T	192	291	381	52	234	338	440
7.4x3.1	8.0x3.5	tall T	172	188	273	50	189	210	273
7.5x4.1	7.3x3.5	LVH	198	94	181	48	236	197	270
7.6x4.3	7.8x3.8	IWI	152	140	224	54	178	157	240
8.0x4.1	8.0x3.5	tall T	100	113	161	28	150	142	224
8.0X3.7	8.1X3.6	LVH	114	137	195	36	129	160	171
8.0x3.7	8.1x3.3	LVH	141	172	248	48	168	180	258
8.2x4.0	7.9x4.5	LWI	174	66	152	52	329	67	178
7.6x4.3	8.1x4.4	tall T	141	170	248	48	169	180	258
8.2x4.2	7.6x4.1	normal	198	94	181	48	236	197	270
8.1x3.8	7.2x4.0	LVH	100	113	161	28	150	142	224
8.0x4.5	7.9x4.1	tall T	156	141	220	48	156	152	228
7.1x4.1	7.5x3.6	IWMI	130	116	180	38	180	172	254
7.0x4.4	7.9x4.1	IWMI	190	172	260	50	180	178	266
6.9x4.1	7.8x3.6	tall T	184	99	135	49	224	133	111
8.0x3.1	8.0x3.0	tall T	111	178	248	48	109	190	258
7.1x4.1	7.7x3.8	normal	94	48	119	52	43	47	86
8.2x3.1	7.8x3.2	AWI	125	163	210	22	148	151	208
8.1x3.3	7.7x4.2	LVH	100	56	114	38	95	51	104
7.7x4.1	6.9x3.9	tallT,LVH	198	193	280	48	188	203	290
6.4x3.4	6.0x3.2	normal	143	130	197	53	180	149	207
8.4x3.6	8.2x3.6	tall T	159	129	212	52	139	102	167
7.8x4.1	7.6x3.3	sine wave	109	190	258	46	106	240	299
7.6x4.1	8.2x3.9	normal	112	137	200	40	95	131	193
7.6x3.6	7.8x4.1	tall T	199	150	228	38	290	120	228
8.4x3.1	8.3x4.2	LVH	160	118	193	43	187	153	230
7.1x3.1	7.7x4.2	LWI	117	107	286	56	102	273	325
7.2x4.3	5.8x4.4	LWI	141	170	248	48	169	180	258
7.7x4.1	8.1x3.6	AWI	158	148	218	38	226	144	233
7.2x3.6	7.7x3.9	wide QRS	216	221	320	56	350	209	328
8.6x3.1	7.2x4.1	IWI	239	100	196	48	161	115	185
7.6x4.6	7.4x4.8	normal	120	112	188	52	110	202	272

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